

## Historic, archived document

Do not assume content reflects current scientific knowledge, policies, or practices.





9622  
28312  
esp. 2

# Research Note

LIBRARY  
SERIAL P 70  
APR 11 1957  
U. S. DEPARTMENT OF AGRICULTURE

UNITED STATES DEPARTMENT OF AGRICULTURE  
FOREST SERVICE

INTERMOUNTAIN FOREST & RANGE EXPERIMENT STATION  
OGDEN UTAH

No. 42

March 1957

## ASPEN LEAF BLIGHT IN THE INTERMOUNTAIN REGION

James L. Mielke  
Division of Forest Disease Research

The fungus Marssonina populi (Lib.) Magn. is the cause of a blight of quaking aspen (Populus tremuloides Michx.) not only in the Intermountain Region but also elsewhere within the range of the host. Very little has been published concerning this pathogen and the disease it causes and most of this is based on observational evidence.

### NOMENCLATURE

The causal fungus is referred to as Marssonina populi throughout this paper in conformity with the nomenclature used in Index of Plant Diseases in the United States (10). It is recognized, however, that further taxonomic study of the fungus is badly needed to clarify both the possible synonymy of the nearly one dozen names that have been offered and the specific host relationships if more than one fungus is actually involved.

### DISTRIBUTION AND HOSTS

Marssonina populi is widely distributed in Europe (4). It is also native to North America where it is likewise widespread (2, 7, 10).

Practically all, if not all, species of Populus are hosts of the pathogen. Boyce (2) reports it as a widespread leaf spot and shoot blight of poplars that is prevalent on aspen in the southern Rocky Mountain region. The fungus is also common on aspen in the Intermountain Region, particularly in Utah, western Wyoming, and southeastern Idaho. Marssonina populi has been collected on narrowleaf cottonwood (Populus angustifolia James) in Utah, but this host appears to be low in susceptibility to the organism.

## LIFE HISTORY

The life history of Marssonina populi is not known and there is no evidence in the meager literature on the fungus that an attempt has ever been made to work it out. It has not even been determined where the spores overwinter.

## EPIDEMIOLOGY

Based on observations, epidemic development of the fungus coincides with spring and summer seasons characterized by abundant rainfall. During dry growing seasons little or no evidence of the fungus may be seen.

Possibly the first report of the fungus in the Intermountain Region is that by Meinecke (6) who conducted observations and studies on aspen there between 1917 and 1920. He mentions that a leaf spot, closely related to Marssonina populi, but differing from it somewhat in spore characters, was exceedingly common in the vicinity of the Great Basin Experiment Station on the Manti National Forest.

Pathological observations have been made on the fungus in the Intermountain Region for some time. Years in which it is known to have been common on aspen over portions of the Region are 1939, 1944, 1948, 1949, 1953, and 1956. In 1948 and 1949 the organism caused severe damage to the species on portions of the Cache National Forest. In 1950, however, this 2-year epidemic subsided and the fungus was not found there.

## SYMPTOMS AND DAMAGE

Marssonina populi not only attacks the leaves of poplars but also causes a blight of the shoots (2, 4, 8). Grove (4) reports it as destructive to young poplar trees in Europe. In this country, Stevens (9) mentions the fungus as injurious to poplars in nurseries.

In the Intermountain Region infected aspen leaves begin turning color occasionally in late July but usually sometime in August. Often the diseased leaves are much smaller than normal. Viewed from a distance the crowns of diseased trees show a light tan or bronzed discoloration. A close inspection of the affected leaves reveals spots of varying sizes that are very irregular in outline. The spots are at first tan or brownish and later turn blackish in color. Bordering these spots the leaf tissues are a yellowish to golden color.

One of the results of heavy infection is premature defoliation. Repeated annual infection reduces increment. In some cases twigs and branches are killed and in others the entire tree may die except for some of the roots. Sprouting from these remaining live roots usually perpetuates the stand to some degree.

Evidently aspen in the Intermountain Region rarely reproduces by seed (1, 3, 5). According to Baker (1), this method of reproduction is of no importance in the management of aspen stands in Utah. Suckering from the roots is the method by which regeneration practically always takes place. As a result of these root sprouts aspen stands are largely composed of clones originating from single parent trees.

Some clones have been found to be highly susceptible to Marssonina populi, some immune or practically so, and others intermediate in susceptibility. Because of this situation extensive stands of aspen in which the fungus is epidemic present a very patchy color pattern in late summer. Foliage of immune clones remains a normal green color, while the highly susceptible clones are bronze in color. The line of demarcation between the two is often sharply defined and striking in appearance. Many gradations in color may be observed representing different degrees of susceptibility to the fungus by the different clones present in the stands. Repeat color photographs of aspen stands taken in epidemic years for the fungus verify the existence of the immune and susceptible clones by showing the constancy of the same patchy color patterns of the diseased stands from year to year in which conditions for infection are favorable.

In 1950 on the Cache National Forest, following the two severe infection years of 1948 and 1949, about 30 aspen trees were felled and dissected. In these trees 50 to 60 percent (estimated) of the twigs and branches had been killed by the fungus during the two epidemic years. Cross sections of the trunks and branches of all these trees showed portions of the heartwood stained a light tan fading into a grayish color. Stained areas were very irregular in outline and appeared to originate at pith flecks and to progress from them towards the center of the trunk or branch. The significance of this relationship, if any, has not been determined. No fungus was found in association with the stained wood and pH tests showed it to be much more acid than the unstained or normal-appearing wood.

## CONTROL

No method of controlling the disease caused by Marssonina populi is known. Clones high in susceptibility to the fungus appear to comprise a relatively small portion of the aspen stands in the region. Consequently this native pathogen should not be considered as the cause of a major disease of aspen, i.e., one that will be damaging and destructive over extensive stands of the tree. Aspen cuttings or root sprouts used for propagation purposes should be taken only from immune clones. Areas occupied by highly susceptible clones, which may range in size up to several acres or more, should be avoided as sites for summer homes, picnic areas, campgrounds, etc. Such areas may be noted and mapped during epidemic years for the fungus.

## LITERATURE CITED

- (1) Baker, F. S.  
1925. Aspen in the central Rocky Mountain region. U. S. Dept. Agr. Bul. 1291, 45 pp.
- (2) Boyce, J. S.  
1948. Forest pathology. Ed. 2, 550 pp. McGraw-Hill Book Co. New York.
- (3) Ellison, L.  
1943. A natural seedling of western aspen. Jour. Forestry 41: 767-768.
- (4) Grove, W. B.  
1937. British stem- and leaf-fungi. II. 406 pp. Cambridge University Press
- (5) Larson, G. C.  
1944. More on seedlings of western aspen. Jour. Forestry 42: 452.
- (6) Meinecke, E. P.  
1929. Quaking aspen: a study in applied forest pathology. U. S. Dept. Agr. Tech. Bul. 155, 33 pp.
- (7) Nordin, V. J.  
1953. A leaf spot disease of aspen. In Bi-Monthly Progress Report, Canada Dept. Agr., Vol. 9, Rept. No. 2, p. 4.
- (8) Pirone, P. P.  
1948. Maintenance of shade and ornamental trees. Ed. 2, 436 pp. Oxford University Press. New York.

- (9) Stevens, F. L.  
1925. Plant disease fungi. 469 pp. The Macmillan Co.  
New York.
- (10) U. S. Department of Agriculture  
1953. Index of plant diseases in the United States. V.  
pp. 809-1192.

